Significance of contrast enhancement in cranial computerized tomography after subarachnoid hemorrhage


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Eighty patients with subarachnoid hemorrhage underwent computerized tomography (CT) scanning before and after administration of Conray contrast medium. Abnormal enhancement was seen in visual evaluation of the CT scans in 26 cases, in the regions bordering the subarachnoid spaces. Abnormal enhancement was associated with a poor clinical condition, angiographic spasm, and a poor outcome. Measurements of absorption values in the thalamus revealed significant increases in density after contrast enhancement in those patients whose scans showed abnormal enhancement in the regions bordering the subarachnoid spaces on visual evaluation. The authors suggest that the abnormal enhancement is parenchymal, in the gyri, and is not "subarachnoid." They suggest that it is due to gyral hyperemia or extravasation of contrast material into the cortex resulting from breakdown of the blood-brain barrier, or a combination of both factors.

KEY WORDS • subarachnoid hemorrhage • intracranial aneurysm • computerized tomography • blood-brain barrier

COMPUTERIZED tomography (CT) scanning is of value in the assessment of cases of subarachnoid hemorrhage (SAH). It may reveal the presence of clot in the subarachnoid spaces, demonstrate the distribution of subarachnoid blood (which may enable prediction of the location of an aneurysm, and may help to identify the causative lesion in cases of multiple aneurysms), and identify complications such as intra-cerebral or subdural hematoma causing mass effect, cerebral infarction, or ventricular dilatation.\(^{3,10,12,17,22,26,33,42}\) The use of post-contrast CT scanning following SAH has received relatively little attention in the literature.\(^{9,10,13,18,20,24,27,45}\) Most reports have stressed the possibility of demonstrating the large arteries in the basal cisterns, and large aneurysms. Abnormal post-contrast enhancement in the region of the basal subarachnoid cisterns has been reported in a few series,\(^{9,13,18,20,27,45}\) but the significance of these findings has not been clarified.

Contrast-enhanced scans have not been part of the routine work-up of patients presenting after SAH at Atkinson Morley's Hospital. Because of the large numbers of SAH patients, however (about 150 new cases per annum), we have been able to analyze a sufficient number of individuals who underwent cranial scanning before and after administration of intravenous contrast material, to assess the significance of abnormal enhancement.

Clinical Material and Methods

The records of all patients admitted between 1977 and 1982 to the Atkinson Morley's Hospital following SAH, confirmed by lumbar puncture, were reviewed. All had undergone cranial CT on admission, but only 80 patients were injected with contrast material. These patients form the basis of this study. The age and sex distribution of the patients is shown in Table 1. All

<table>
<thead>
<tr>
<th>TABLE 1</th>
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<tbody>
<tr>
<td><strong>Age and sex distribution of patients</strong></td>
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<tr>
<td><strong>Age (yrs)</strong></td>
</tr>
<tr>
<td>18–29</td>
</tr>
<tr>
<td>30–39</td>
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<tr>
<td>40–49</td>
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<tr>
<td>50–59</td>
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<tr>
<td>60–69</td>
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</table>
scans were performed within 5 weeks of the most recent SAH. Angiography was carried out in 75 cases. The distribution of the causative aneurysms is shown in Table 2. The CT scanning was performed on an EMI 1010 scanner and the scans were displayed on a 160 × 160 matrix. Contrast enhancement was effected by the intravenous injection of 50 ml of sodium iothalamate (Conray 420) over a period of 2 to 3 minutes. Scanning was performed within 5 days of the most recent hemorrhage in 45 patients, and in 35 patients between 6 and 20 days following SAH. The original scans were reviewed by a neuroradiologist to establish the presence or absence of abnormal contrast enhancement (abnormal increased density following the administration of Conray) by "visual evaluation." Particular attention was paid to the areas bordering the basal subarachnoid spaces. In those patients whose enhanced scans showed blood in the subarachnoid spaces (increased attenuation of blood density in the basal cisterns or in the subarachnoid spaces of the fissures and sulci), the scans were assessed for visual evidence of additional areas of increased soft-tissue density following intravenous administration of contrast medium.

Of the 80 patients whose scans were assessed visually, the original tape-stored data of 43 were suitable for use. These data were reconstructed on a graphic display system, and the mean absorption values of 50-sq mm areas (considered to be the most convenient area to ensure homogeneity) were measured (absorption measurements) using the region of interest (ROI) technique. The mean absorption value of a chosen area was considered to be representative (that is, homogeneous) only if the standard deviation (SD) of the mean value did not exceed 4.5 Hounsfield units (HU). The baseline values of normal post-contrast enhancement were determined in 14 patients presenting with complaints of headache, depression, and obsessional neurosis, whose neurological examinations were normal, and whose CT scans were also considered to be normal. The greatest possible care was taken to compare corresponding brain areas when the measurements of absorption were determined before and after administration of contrast material.

The extent of blood in the subarachnoid spaces shown in the scans was classified according to the anatomical location of the subarachnoid blood clot: in Group 1 no subarachnoid blood clot; in Group 2 blood clot was visible in one or two cisterns and/or fissures; in Group 3 blood clot was visible in three or more cisterns and/or fissures; in Group 4 there was

<table>
<thead>
<tr>
<th>Location of Causative Aneurysm</th>
<th>No. of Cases</th>
<th>Abnormal Enhancement</th>
<th>No Abnormal Enhancement</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td></td>
<td>≤ 5 Days Post-SAH</td>
<td>&gt; 5 Days Post-SAH</td>
</tr>
<tr>
<td>anterior communicating artery</td>
<td>20</td>
<td>6</td>
<td>2</td>
</tr>
<tr>
<td>middle cerebral artery</td>
<td>34</td>
<td>8</td>
<td>7</td>
</tr>
<tr>
<td>internal carotid artery including posterior communicating artery</td>
<td>17</td>
<td>2</td>
<td>2</td>
</tr>
<tr>
<td>vertebral/basilar artery</td>
<td>3</td>
<td>--</td>
<td>--</td>
</tr>
<tr>
<td>source of SAH not found (negative angiograms) or not verified (early death)</td>
<td>6</td>
<td>2</td>
<td>--</td>
</tr>
</tbody>
</table>

* SAH = subarachnoid hemorrhage.

FIG. 1. Computerized tomography scans taken within 5 days of a subarachnoid hemorrhage in a patient whose unenhanced scans (left pair) showed no subarachnoid blood. The post-contrast scans (right pair) showed enhancement in the regions bordering the basal subarachnoid spaces, the fissures, and the sulci.
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localized blood clot extending into the ventricles or brain parenchyma, or diffuse subarachnoid clot filling not only the cisterns but also the sulci of the hemispheres.

Arterial spasm detected on angiography was classified as follows: 1) no spasm; 2) moderate spasm (slight decrease in arterial diameter of a related vessel); 3) marked spasm (decrease of arterial diameter of 50% or more in one or more of the major vessels related to the aneurysm); 4) diffuse spasm (50% or more narrowing of several major vessels).

The clinical condition of the patients was graded according to the classification of Hunt and Hess. The outcome was described as excellent when the patient had no residual deficit; good when minor deficits did not prevent a normal life; and disabled when the patient was dependent on help due to major deficits. Fatal outcome was classified as due to a rebleed, cerebral infarction, or other cause. Student’s t-test and the chi-square test were used for statistical analysis.

Results

Of the 45 patients who were scanned within 5 days of hemorrhage (Table 3), increased density (attenuation) was observed in 18 in the regions bordering the basal subarachnoid spaces on the post-contrast scan ("abnormal enhancement"). Increased density was not confined to the regions bordering the basal cisterns although it was most prominent in that location. Abnormal enhancement was also seen bordering the interhemispheric and Sylvian fissures and the cerebral sulci (Figs. 1, 2, and 3).

Of the 35 patients whose CT scan was obtained later than the 5th post-hemorrhage day (Table 3), eight had areas of low density on the pre-contrast scan which were attributable to infarction. The post-contrast enhancement in those cases was confined to the infarcted areas

<table>
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<th>TABLE 3</th>
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<tr>
<td>Abnormal enhancement and time interval from SAH to computerized tomography</td>
</tr>
<tr>
<td>Findings</td>
</tr>
<tr>
<td></td>
</tr>
<tr>
<td>enhancement</td>
</tr>
<tr>
<td>no enhancement</td>
</tr>
<tr>
<td>total cases</td>
</tr>
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</table>

FIG. 2. Same case as illustrated in Fig. 1. Computerized tomography scans showing areas of absorption measurements (outlined in white squares). Left: Unenhanced scan. Right: Scan after contrast administration.

FIG. 3. Computerized tomography scans taken within 5 days of a subarachnoid hemorrhage in a patient whose unenhanced scans (left pair) showed a subarachnoid blood clot in the interhemispheric and Sylvian fissures, in the basal cisterns, and in the sulci. The enhanced scans (right pair) showed additional areas of increased density (abnormal enhancement).
only. Of the 27 patients whose unenhanced scans later than the 5th day did not show low density, three showed post-contrast enhancement. Although post-contrast enhancement was most obvious on visual inspection in the regions bordering the basal subarachnoid spaces and major fissures, the measurements of absorption values from these areas showed very high SD values; not only in the subjects of the study, but also in the control scans. The close proximity of cerebrospinal fluid (CSF), blood vessels, blood, and cerebral cortex renders these areas non-homogeneous and the absorption values unsuitable for analysis. The area of the basal ganglia (thalamus) was found to be the most homogeneous, having low SD values (less than 4.5 HU). The data were therefore analyzed by grouping of patients according to the presence or absence of contrast enhancement on visual evaluation in the regions bordering the basal subarachnoid spaces, and comparing the difference in mean absorption values measured in the basal ganglia before and after contrast enhancement in each group. In the control scans, the increase in attenuation following contrast enhancement was 1.2 HU (SD = 1.4; that is, not appreciable).

Table 4 shows that a statistically significant increase in attenuation was observed only in the group of patients who were scanned within 5 days of hemorrhage who had abnormal enhancement in the regions bordering the subarachnoid spaces as detected by visual evaluation (p < 0.01). Abnormal enhancement was more frequent in the poorer clinical grades (p < 0.01), and the outcome in these patients was less favorable (p < 0.01, Table 5). Of the 17 patients who did not have detectable subarachnoid clot when scanned within 5 days of hemorrhage, none had abnormal enhancement. Of the 28 patients whose scans showed subarachnoid clot when taken within 5 days, 18 developed abnormal enhancement (p < 0.01, Table 6). Systemic arterial hypertension was associated with a statistically significant increase in the incidence of abnormal enhancement (p < 0.01, Table 6).

Spasm was not seen on any angiogram obtained within 5 days of hemorrhage. In the patients whose angiography was performed more than 5 days after hemorrhage, there was a significant correlation (p < 0.01) between the presence of spasm and abnormal post-contrast enhancement on visual evaluation of the CT scan (Table 7). There was a correlation between the amount of subarachnoid clot and the clinical condition of the patients (Table 8). Whereas 13 of 17 cases without detectable subarachnoid blood were in favorable clinical condition (Grades I and II), 19 of 28 patients with detectable subarachnoid clot on the unenhanced CT scans were in the less favorable clinical grades.
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TABLE 6
Abnormal enhancement on CT correlated with amount of subarachnoid blood clot and blood pressure*

<table>
<thead>
<tr>
<th>Factors</th>
<th>Abnormal Enhancement ≤ 5 Days Post-SAH</th>
<th>&gt; 5 Days Post-SAH</th>
<th>No Abnormal Enhancement ≤ 5 Days Post-SAH</th>
<th>&gt; 5 Days Post-SAH</th>
</tr>
</thead>
<tbody>
<tr>
<td>extent of subarachnoid blood clot</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>none</td>
<td>0</td>
<td>5</td>
<td>17</td>
<td>21</td>
</tr>
<tr>
<td>1 or 2 cisterns or fissures</td>
<td>8</td>
<td>4</td>
<td>4</td>
<td>2</td>
</tr>
<tr>
<td>3 or more cisterns or fissures</td>
<td>4</td>
<td>0</td>
<td>3</td>
<td>0</td>
</tr>
<tr>
<td>extension into ventricles or brain</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>parenchyma, or diffuse SAH blood</td>
<td>6</td>
<td>2</td>
<td>3</td>
<td>1</td>
</tr>
<tr>
<td>pressure</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>≤ 140/90 mm Hg (normal)</td>
<td>8</td>
<td>6</td>
<td>20</td>
<td>16</td>
</tr>
<tr>
<td>between 140–200/90–120 mm Hg</td>
<td>8</td>
<td>4</td>
<td>6</td>
<td>5</td>
</tr>
<tr>
<td>over 200 mm Hg (known hypertensive)</td>
<td>2</td>
<td>1</td>
<td>1</td>
<td>3</td>
</tr>
</tbody>
</table>

* Patient's blood pressure was assessed during the course of hospitalization. CT = computerized tomography; SAH = subarachnoid hemorrhage.

A correlation between abnormal enhancement, clinical condition, and the outcome of the disease is shown in Table 9. Patients in Hunt and Hess Grades I and II generally had a favorable outcome uninfluenced by abnormal enhancement (the Grade II patient whose condition was considered "disabled" suffered a middle cerebral artery occlusion during surgery, and hemiplegia was manifest immediately postoperatively). Of the 16 patients in Hunt and Hess Grade III, nine had abnormal enhancement. When deaths secondary to rebleeding or other causes are excluded, the outcome of seven patients whose scans showed enhancement and of six patients whose scans showed no enhancement can be compared. Four of the patients with enhancement died from cerebral infarction, but all six without enhancement had favorable outcome.

TABLE 7
Relationship between degree of spasm on angiography and abnormal enhancement on CT scan*

<table>
<thead>
<tr>
<th>Degree of Spasm on Angiography</th>
<th>Abnormal Enhancement ≤ 5 Days Post-SAH</th>
<th>&gt; 5 Days Post-SAH</th>
<th>No Abnormal Enhancement ≤ 5 Days Post-SAH</th>
<th>&gt; 5 Days Post-SAH</th>
</tr>
</thead>
<tbody>
<tr>
<td>angiography &gt; 5 days</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>post-SAH</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>no spasm</td>
<td>1</td>
<td>6</td>
<td>12</td>
<td>19</td>
</tr>
<tr>
<td>moderate spasm</td>
<td>1</td>
<td>0</td>
<td>4</td>
<td>3</td>
</tr>
<tr>
<td>marked spasm</td>
<td>4</td>
<td>3</td>
<td>3</td>
<td>1</td>
</tr>
<tr>
<td>severe &amp;/or diffuse spasm</td>
<td>3</td>
<td>2</td>
<td>0</td>
<td>1</td>
</tr>
<tr>
<td>angiography not performed</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>angiography ≤ 5 days</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>of SAH</td>
<td>5</td>
<td>0</td>
<td>3</td>
<td>1</td>
</tr>
</tbody>
</table>

* CT = computerized tomography; SAH = subarachnoid hemorrhage.

TABLE 8
Extent of blood clot in subarachnoid space correlated with clinical condition of patient*

<table>
<thead>
<tr>
<th>Extent of Subarachnoid Blood Clot</th>
<th>Clinical Grade</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>I</td>
</tr>
<tr>
<td>none</td>
<td>7</td>
</tr>
<tr>
<td>1 or 2 fissures or cisterns</td>
<td>1</td>
</tr>
<tr>
<td>3 or more fissures or cisterns</td>
<td>0</td>
</tr>
<tr>
<td>extension into ventricles or</td>
<td>0</td>
</tr>
<tr>
<td>brain parenchyma, or diffuse SAH</td>
<td></td>
</tr>
</tbody>
</table>

* Extent of blood clot assessed on computerized tomography within 5 days of subarachnoid hemorrhage (SAH). Clinical status according to the Hunt and Hess classification.*

Discussion

Abnormal tissue enhancement on cranial CT scans has been reported as a consequence of SAH.61,13,18,20,27,45 In our series 36% of the 80 patients who were scanned within 3 weeks of SAH demonstrated this phenomenon. Eighteen of the 45 patients who were scanned within 5 days of the most recent bleed had abnormal parenchymal enhancement. Eleven of the 35 patients who were scanned more than 5 days after hemorrhage had abnormal enhancement, which was confined to infarcted areas in eight cases. We recognize that this may not represent the true incidence, as only a small proportion of the total number of patients admitted under our care following SAH were given intravenous contrast medium. Enhanced scanning was performed in an attempt to identify aneurysms in those patients scanned within 5 days, and also to identify infarction in those patients scanned later. In those patients scanned within 5 days of hemorrhage the enhancement was present in the regions bordering the basal cisterns, and often also the perimesencephalic cisternal spaces, the interhemispheric.
spherical and Sylvian fissures, and the sulci of both hemispheres. In two cases, the examination was performed both within and after 5 days of hemorrhage. In the acute stage (under 5 days), bilateral generalized soft-tissue enhancement was observed in both cases, but the unenhanced scans in the chronic phase showed infarction which enhanced following administration of intravenous contrast medium.

Intravenous contrast medium is normally confined within the blood-brain barrier, so that normal brain substance increases in attenuation by only a low amount proportional both to the concentration of iodine within the blood stream and to the volume of blood within the brain substance. Our measurements in patients who had not sustained a hemorrhage and who had normal scans were in keeping with published values.

Some authors have described abnormal enhancement on CT scanning as “subarachnoid.” We consider the term inappropriate, implying as it does the enhancement of tissues or structures within the subarachnoid space, which do not normally enhance. We believe that the abnormal enhancement is present in the gyri, as previously suggested, which border the subarachnoid space; thus, the enhancement is parenchymal. The partial-volume effect may have misled those authors who reported “subarachnoid” enhancement. It has been suggested that the “subarachnoid” enhancement may be due to leakage of contrast medium into the subarachnoid cisterns from breakdown of the blood-brain barrier. "Meningeal hyperemia" has also been suggested.

We believe that increased blood volume in the small vessels of the cerebral cortex may play a part in the abnormal enhancement. There is evidence of paralysis of small-vessel autoregulation which leads to dilatation of arterioles, capillaries, and venules, with resultant increase in cerebral blood volume following SAH both in man and in animal models. Major impairment of small-vessel autoregulation leading to massive intraparenchymal dilatation of small vessels has been particularly reported in patients with severe or generalized vascular spasm, and in patients in clinical Grades III and IV of Hunt and Hess.

We believe that extravasation of contrast medium into the interstitial fluid of the cortical gray matter resulting from impairment of the blood-brain barrier may also play a part in the abnormal enhancement. The very considerable increase in enhancement in several of our cases (Table 4) offers evidence that extravasation of contrast medium (that is, abnormal capillary permeability) is a factor in abnormal post-SAH enhancement. An increase in cerebral blood volume by a factor in excess of 3 would be required to account for the increase in absorption measurements if the enhancement were due entirely to hyperemia of the gyri.

Contrary to previous reports which have suggested that there is no correlation between abnormal postcontrast enhancement and clinical condition, our series shows that patients with abnormal enhancement are likely to be in less favorable clinical grades, have a high incidence of marked or diffuse spasm, have a poorer outcome independent of surgical or conservative treatment, and develop cerebral infarction more frequently. Correlation between “subarachnoid” enhancement and neurological deficit has been recently suggested, but a high incidence of hydrocephalus requiring shunting was present in that series, which complicates interpretation of the significance of the data. Hydrocephalus requiring treatment was not a feature in our series.

Systemic arterial hypertension was associated with an increased incidence of abnormal enhancement. The greater tendency to loss of autoregulation in hypertensive patients following SAH supports the theory that abnormal enhancement is related to cortical hyperemia. The association of extensive subarachnoidal clot with the development of abnormal enhancement suggests that vasoactive substances arising from the breakdown of blood clot may play a part, a possibility supported by the fact that enhancement is most obvious in the cortical areas bordering the major subarachnoidal spaces.

Although the contrast enhancement is most obvious in the cortex, particularly in the neighborhood of the subarachnoidal spaces, the absorption measurements in the basal ganglia suggest that the phenomenon is prob-
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ably widespread throughout the brain. The thalamic enhancement, whether due to hyperemia or extravasa-
tion or both, probably also occurs in the hypothalamus,
which may play a part in the hypothalamic syndromes
that have been reported following SAH.8,11

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